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THE PLANT DISEASE REPORTER

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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IN THIS ISSUE

Observations that may help to explain the distribution of cucurbit viruses in central Florida are reported by C. W. Anderson, page 377; the viruses concerned are the watermelon and cucumber mosaic viruses, a mosaic virus from the wild plant Melothria, and another from cantaloupe resembling the South Carolina cantaloupe mosaic virus.

Warid A. Warid and A. G. Plakidas, page 380, report exceptional heat tolerance for some viruses occurring naturally on cowpea in Oklahoma and Louisiana.

From hydrangea plants affected with a newly observed disease termed rugose mosaic, a ringspot virus of unknown identity was isolated, according to Philip Brierley and Floyd F. Smith, page 382.

Paul D. Keener reported occurrence of Darluca filum on Puccinia sorghi in Pennsylvania in 1933; numerous other rusts were infected with D. filum isolated from P. sorghi, page 384.

Natural infection of chestnut oak by Chalara quercina is reported from Pennsylvania by Charles L. Fergus, page 386.

T. L. Guyton, page 386, reports that a large proportion of oak trees blazed in April to mark a trail in Pennsylvania became infected by Chalara quercina and were killed by the middle of September.

The Sclerospora disease of sugarcane, recently reported in Peru, should not become of great importance provided field control measures are applied, according to E. V. Abbott and J. P. Martin, page 387.

C. E. Yarwood and H. T. Harvey report California tests proving the truth of growers' reports that washing cherries with water after picking improved keeping, page 389.

Severe winter injury to trees in Illinois, mostly attributed to the sudden freeze in November 1951, is reported by Paul F. Hoffman, page 389.

P. A. Young's report on diseases observed in west Texas is concerned mostly with curly top on various vegetable crops, page 390.

New records of plant diseases, page 391, include reports on take-all on wheat in Georgia, by H. B. Harris and others, and Fusarium wilt of flue-cured tobacco in Virginia, by Wilbert A. Jenkins.

A correction, page 391.

August weather, page 392.

THE DISTRIBUTION OF CUCURBIT VIRUSES IN CENTRAL FLORIDA[1]

C. W. Anderson

Cucumber mosaic (CM) and watermelon mosaic (WM) viruses have been reported previously from central Florida. Recently, variations in symptoms produced by certain isolates believed to be WM virus were noted. This report presents some ideas which may explain the distribution of those viruses in Florida.

Preliminary tests of some of the isolates considered to be in the WM group indicated that their physical properties were close to or identical with those of typical WM virus. In repeated tests one such isolate invariably caused symptoms different from those of typical WM in several different cucurbits.

METHODS

Observations were made for virus infection in a large number of cucurbit fields and virus was successfully transmitted to test plants from naturally infected cucurbits from 48 fields. WM virus induces systemic mosaic symptoms without primary lesions in cucumber, squash, and watermelon, but does not infect pepper or tobacco. CM virus isolates caused primary lesions in the three cucurbits, systemic symptoms in cucumber and squash and often in watermelon, and systemic mosaic symptoms in pepper and tobacco. In all but two cases watermelon plants, straight-neck squash or cucumber plants, or both, and pepper or tobacco plants, or both, were inoculated with each virus isolate. The two excepted isolates were identified by inoculation to less than three of the above plant species. The carborundum method of inoculation was used and one control plant was kept for each test plant. All controls remained healthy.

RESULTS

Fifty-two virus isolates from 48 cucurbit fields were classified by means of the inoculations described above. The counties and hosts from which the isolates were obtained are also indicated in Table 1. Data in this table show that WM virus was most common in the Manatee-Hardee-Hillsborough and Alachua-Marion County areas and in yellow crook-neck squash. CM virus was most common in the Orange-Seminole-Volusia County area and in cucumber. Crook-neck squash was commonly grown in much of the first two areas, but not in the third. Cocozelle and straight-neck squashes were often planted in parts of Seminole County and cucumbers in Orange County. WM fruit symptoms in yellow squashes are especially striking. Fruit symptoms of both viruses are similar in cucumber, but those of CM are stronger. WM does not kill the plants in the striking manner that CM does.

Table 1. Classification of 52 isolates from 48 cucurbit fields by means of host reactions.

Source: Locality (Florida Counties)	Virus WM	Virus CM	Source: Natural Host	Virus WM	Virus CM
Manatee-Hardee-Hillsborough	18	3	Crook-neck squash	19	0
Orange-Seminole-Volusia	1	10	Other squash	5	5
Sumter	1	1	Cucumber	5	9
Alachua-Marion	14	1	Cantaloupe	4	1
South Florida	2	0	Watermelon-Citron	3	0
Totals*	36	15	Totals	36	15

* The 52nd isolate, obtained from Marion County cantaloupe, did not fit either virus and is discussed later.

[1]Florida Agricultural Experiment Station Journal Series, No. 82.

Data on natural infection of other plant species with cucurbit viruses are being accumulated, but only preliminary results concerning Commelina spp. and Melothria spp. will be presented here. The writer is indebted to E. West for determinations of Commelina spp.

Anderson (2) briefly reviewed the history of cucurbit viruses in Florida, mentioning that natural infection of Melothria spp. by a squash virus was reported from southern Florida by Felix. Naturally infected Melothria spp. were not then known in central Florida. Recently, WM virus was isolated from naturally infected Melothria spp. in Manatee, Seminole, and Alachua Counties. To date, very large infected Melothria plants and high percentages of WM-infection of Melothria have been noted only in Palma Sola, Manatee County. Here, also, infected Melothria grew much closer to cultivated cucurbits than was the case at Sanford, Seminole County. Moreover, the amount of WM infection of cultivated cucurbits at Palma Sola where crook-neck squash was the most common cucurbit planted, was higher than that found elsewhere, regardless of the type of cucurbit grown. A number of crook-neck squash fields were observed in localities where no Melothria plants were found. Often the squash plants were healthy.

CM virus was isolated from Commelina gigas Small (1), C. nudiflora L. (C. longicaulis Jacq.) (7), and C. elegans H.B.K. C. nudiflora is the most important host. Wellman's work (7) with C. nudiflora in Seminole County is well known. In Manatee and Hillsborough Counties Commelina spp. were found within a few feet of WM-infected squash fields, but no CM was noted in the fields and an attempt to transmit a virus from apparently diseased Commelina to the cucurbits failed. This may indicate that species of Commelina carry more than one virus in Florida, as Townsend and Felix (6) suggested.

WM seems to spread more rapidly in squash than does CM and, in one or two laboratory tests, older squash plants seemed resistant to CM but not to WM. Wellman (7), however, reported especially rapid spread of CM in squash, and his illustrations seem to represent CM infection. In some areas (e.g., Orange County) cucumbers were planted in fields surrounded by citrus groves and CM was prevalent but WM was not. Commelina spp. were found in both the cucumber fields and adjacent groves. Although Melothria sometimes grows on uncultivated ditch banks in citrus areas, the only wild cucurbit yet found in citrus groves is Momordica charantia, a species reported by Anderson (2) to be nonsusceptible to WM virus.

Once greenhouse-grown watermelon plants infected with WM were planted outdoors and symptom severity greatly decreased at times. The naturally infected watermelon plants from which WM virus was isolated showed only mild symptoms, although severe symptoms do occur.

DISCUSSION

The data are considered insufficient to warrant definite conclusions, but they do afford tentative explanations for several of the observations as follows:

Melothria spp. are important sources of WM virus. Although infected Melothria was not found wherever WM occurred, it is especially common where WM was most prevalent.

Anderson (2) reviewed the reports of seedborne cucurbit viruses in California and Michigan and Rader et al. (5) reported such a virus in New York. Although there is yet no evidence that WM is seedborne (2), the possibility cannot be entirely discounted. Moreover, any aphid-transmissible seedborne cucurbit virus could readily establish itself if it could infect Melothria or, perhaps, Momordica. The relative absence of cucumber beetles among central Florida cucurbit fields may explain the apparent absence of seedborne beetle-transmitted viruses such as squash mosaic (4).

The apparent association between Yellow Summer Crook-neck squash and WM and between cucumber and CM may be explained as follows: Symptom differences between the two viruses are such that CM in cucumber and WM in yellow squash are commonly brought to our attention, while WM infection of green-fruited cucurbits, including even Cocozelle squash, is overlooked. An additional reason for the association of WM with crook-neck squash is that different farming areas specialize in different cucurbits. For instance, crook-neck squash is common in Palma Sola, Manatee County, while other areas specialize in cucumber or watermelon and Cocozelle and straight-neck squash are more commonly grown in Sanford, Seminole County. Since the data indicate striking differences in prevalence of the two viruses in different areas, and since Melothria and Commelina evidently do not commonly grow in the same situations, a correlation between the type of cucurbit grown in an area and the particular virus with which it is associated is to be expected. Watermelon, especially, deserves further investigation to determine the prevalence of cucurbit viruses in this crop. There seems no reason why WM should not be common in this plant, provided infected Melothria occurs in the watermelon areas. The rarity of such reports may be accounted for because of the green fruit of the watermelon, the possible prevalence of milder strains of WM virus, and the partial masking of leaf symptoms under some condi-

tions, rather than from actual absence of the virus.

If WM does spread more rapidly than CM in squash, rapid CM spread may be correlated with special conditions. These may include the planting of squash following incompletely eradicated diseased celery, the presence of large aphid populations on neighboring crucifers, etc., and proximity of squash to diseased Commelina.

Despite the less evident WM symptoms of green-fruited cucurbits, WM probably damages all its hosts through yield reduction and loss of marketability.

The unusual isolate obtained from Marion County cantaloupe somewhat resembled the cantaloupe mosaic virus reported from South Carolina by Aycock (3) in preliminary tests of its host reactions. Although Aycock kindly supplied pictures of the symptoms of his virus in several hosts, it cannot yet be definitely stated that these two viruses are related to each other. Neither Melothria nor Commelina was noted in the vicinity of the Marion County field.

A fourth virus type, Melothria mosaic (MM), was obtained from Manatee County. It differs from WM and CM viruses and from the Marion County cantaloupe virus in symptoms, host range, and physical properties. The reason for the absence of MM virus in cultivated squash fields is unknown. Possibly the virus is not aphid-borne and its vector rarely attacks cultivated cucurbits, or it may have been undetected in complexes with WM or CM viruses. If it were common, it should have been obtained alone from cultivated squash.

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FLORIDA AGRICULTURAL EXPERIMENT STATION, GAINESVILLE

THERMOPHILIC VIRUSES NATURALLY INFECTIOUS TO COWPEA

Warid A. Warid and A. G. Plakidas[1]

Apropos of Brierley's (1) recent paper on the exceptional heat tolerance of the virus of chrysanthemum stunt, we wish to report a similar experience with a number of viruses naturally occurring on cowpea (Vigna sinensis Endl.).

A large number (180 in all) of cowpea plants of different varieties showing symptoms suspected of being of virus nature were collected in Oklahoma and Louisiana. By inoculating differential hosts (31 varieties of cowpea and 10 other plants, namely, cucumber, soybean, alfalfa, bean, lima bean, broadbean, pea, pepper, Turkish tobacco, and Nicotiana glutinosa L.), four distinct single viruses and three virus complexes were determined to be present in this collection. For convenience, these are designated in this discussion as V.V. (Vigna virus) 1, V.V.2, V.V. 3, V.V. 3A, and C. (Complex) 1, C. 2, and C. 3.

In attempts to determine the thermal inactivation points of these viruses, considerable difficulties were encountered and results which were unexpected and unusual were obtained. Infected plants were ground in a meat chopper and the juice was extracted by squeezing the ground pulp through two layers of cheesecloth. The crude juice was diluted with an equal volume of distilled water, and the diluted juice was placed in thin-walled, stoppered glass tubes, 2 cc. to each tube. The tubes were immersed in an electrically heated, stirred water bath. The temperature of the bath was controlled by a thermostat, and it remained constant at the desired temperature within a half degree C. It was found that it took approximately 4 minutes for the juice inside the tube to attain the temperature of the water bath; so for a 10-minute exposure to a particular temperature, the tubes were actually kept in the bath for 14 minutes, after which they were immediately plunged in ice water. The cooled juice was then used to inoculate cowpea plants (Blackeye variety).

Viruses V.V. 2 and V.V. 3A were inactivated by a 10-minute exposure to 98° - 100° C. The others were still infectious after 10-minute exposures to 100° C. (the highest temperature of the water bath). As these results seemed unusual, the temperature of the water bath was rechecked and the experiment repeated. Similar results were obtained.

A mineral oil (Marcol) bath was then substituted for the water bath and its temperature was raised at intervals of 4° C. to 108° C. Viruses V.V. 3, C. 2, and C. 3 were inactivated between 100° and 104° C. and V.V. 1 between 104° C. and 108° C., but C. 1 was still infectious after a 10-minute exposure to 108° C. The juice actually boiled in the oil bath. In the first test, in which the tubes were placed in the oil bath tightly stoppered, the stoppers blew off and the juice blew to the ceiling of the room. In subsequent tests, the stoppers were loosened to allow the escape of steam from the boiling juice. The actual temperature of the boiling juice was not determined. It was probably slightly above that of boiling water because of solutes in the crude juice. The results of a representative experiment are shown in Table 1.

Table 1. Comparison of the thermal inactivation points of 4 single viruses and 3 virus complexes naturally infectious to Vigna sinensis. The Blackeye variety of cowpea was used as the test plant. The numerator indicates number of plants inoculated and the denominator number of plants infected.

Temperature of oil bath (° C.)	Virus						
	V.V. 1	V.V. 2	V.V. 3	V.V. 3A	C. 1	C. 2	C. 3
Check; not heated	10/10	10/10	10/10	10/10	10/10	10/10	10/10
90	8/8	8/8	8/8	8/8	8/8	8/8	8/8
94	8/8	8/4	8/8	8/6	8/8	8/8	8/8
98	8/7	8/2	8/8	8/2	8/8	8/5	8/8
100	8/4	8/0	8/5	8/0	8/8	8/2	8/4
104	8/1	8/0	8/0	8/0	8/6	8/0	8/0
108	8/0	8/0	8/0	8/0	8/3	8/0	8/0

[1]Formerly graduate student in the Department of Botany, Bacteriology and Plant Pathology, Louisiana State University, now plant breeder with Egyptian Ministry of Agriculture, Cairo, Egypt; and Professor of Botany and Plant Pathologist, Louisiana State University, respectively.

These cowpea viruses were described in a Master's thesis (2), a copy of which is deposited in the library of the Louisiana State University, and a brief abstract (3) was published in Phytopathology. The data relative to the exceptional heat tolerance of these viruses were withheld pending further work, but as there is no prospect of continuing work on this subject in the foreseeable future, the data are now presented in the hope that they might be of interest to other investigators.

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A RINGSPOT VIRUS FROM HYDRANGEA INFECTIOUS TO HERBACEOUS PLANTS

Philip Brierley[1] and Floyd F. Smith[2]

Several types of disease with symptoms suggestive of virus etiology have appeared in florists' hydrangeas during the last five years. In February 1947 a die back with brown canker-like lesions in stems and chlorosis and rugosity of leaves caused considerable damage in the varieties Merveille, Rosabelle, and Strafford in a greenhouse in eastern Pennsylvania. The grower stated that the disease had been seen in previous seasons, but never before in destructive form. Inarch grafting reproduced die back symptoms in apparently healthy commercial plants of the Merveille variety after four months. No further outbreaks of this disease have come to our attention since 1947 and some of the samples collected in that year have been retained in the greenhouse at Beltsville without further expression of die back symptoms.

Broad light green rings, 1 cm. or less in diameter, are not uncommon in mature leaves of hydrangeas, and large yellow or brown rings, as much as 5 cm. in diameter, are less frequently seen. Mosaic-like patterns, with pale green blotches irregularly distributed along the branch veins, are not uncommon. As the lower leaves age and lose their green color, the brown pigments may become rearranged to form ring or watermark patterns. None of these symptoms appear to be associated with a marked reduction in vigor of the affected hydrangeas or quality of the flowers. Several attempts to culture in tobacco a virus from hydrangea leaves with symptoms of one of these types have failed. One inoculation in December 1948 from Strafford hydrangeas showing broad light green rings yielded white ring primary lesions in tobacco, but no systemic symptoms followed and the virus was not successfully subcultured.

In May 1952, a North Carolina florist brought to our attention a disease in Strafford hydrangeas that rendered them unsalable. The flowers were small and poorly developed and the leaves stiff, rugose, and mottled light and dark green. Vegetative side shoots developed after the flowering stems were cut also bore stiff rugose leaves at first, but by July the younger leaves were nearly normal although smaller and narrower than those of healthy plants. From such plants affected with the disease here termed rugose mosaic, a ringspot virus was readily isolated in tobacco with the aid of carborundum and the usual manual technique.

Turkish tobacco var. Samsun developed white necrotic ring primaries 8 days after inoculation from hydrangeas. Rings were large, usually about 1 cm. in diameter, with single or double narrow boundary lines. Systemic symptoms developed slowly as similar but multiple rings or target marks. The upper leaves remained unmarked but the virus could be demonstrated in these by subtransfer. The virus was transmitted from tobacco to cucumber, zinnia, China aster, and snapdragon. Cucumber var. A. & C. expressed solid yellow spot primaries in cotyledons after 5 days, a systemic yellow vein mosaic in 7 days, mild yellowish mottling in 18 days, and recovery in 20 days. Zinnia showed a systemic yellow vein mosaic after 6 days and recovered after 14 days. China aster developed a systemic yellow mottle after 11 days and a brown line etch pattern locally after 18 days. No symptoms were detected in snapdragon but the virus was recovered in tobacco from this species. Plants of a number of other species inoculated appear to be insusceptible.

The identity of the hydrangea virus is not known. It failed to protect tobacco from tobacco ringspot virus and failed to protect zinnia from cucumber mosaic virus. The role of this virus in producing symptoms in hydrangea cannot yet be defined.

Common pests of hydrangea in the greenhouse include the meadow spittle bug, Philaenus leucophthalmus (L.) and four species of aphids, the potato, Macrosiphum solanifolii (Ashm.), crescent-marked lily, Myzus circumflexus (Buck.), green peach, M. persicae (Sulz.), and foxglove, M. solani (Kltb.). Tests were made in 1947 with these insects as possible vectors of hydrangea die back virus. The aphids were starved for 2 hours, fed for 15 minutes on source plants, and then allowed to feed for one or more days on healthy hydrangea plants. The spittle bugs were fed on source plants for three days before transfer. In these tests, summarized in Table 1, die back was not transmitted in the non-persistent manner by the 4 species of aphids or in a feeding period of 18 days by the meadow spittle bug.

Three species of aphids were present on the samples of hydrangea rugose mosaic when

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Table 1. Tests with 5 species of insects which failed to transmit a virus from die back hydrangea to hydrangea.

Insect species	Insects in test	Infection period	Plants exposed
	number	days	number
Macrosiphum solanifolii	75	4	5
Myzus circumflexus	75	3	5
M. persicae	300	1-6	15
M. solani	125	9	5
Philaenus leucophthalmus	75	18	10

collected in North Carolina in May 1952. In tests thus far conducted there is no evidence of transmission of a virus from the rugose mosaic source plants to hydrangea seedlings by the aphids Myzus circumflexus, M. persicae, or M. solani, but no final conclusions can yet be drawn.

We are indebted to Prof. C. B. Link of the University of Maryland for supplying hydrangea seedlings for this study.

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING AND BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE

THE OCCURRENCE OF DARLUCA FILUM (BIV.) CASTAGNE ON PUCCINIA SORGHI SCHW.

Paul D. Keener

Semeniuk and Vestal (3) in a recent report concerning leaf diseases of corn refer to the literature relating to various rust hosts of the hyperparasite Darluca filum (Biv.) Castagne in the United States and Canada. In discussing the occurrence of D. filum on various uredineous hosts, the assertion was made (Page 173, bottom) that the fungus has not been previously reported on corn rust (".....but not hitherto on corn rust".) (3).

Keener (1) recorded the occurrence of D. filum on corn rust (Puccinia sorghi Schw.) in Pennsylvania as early as 1933. (Fig. 1). The hyperparasite was isolated into pure culture by monosporic macroconidial procedures on lima bean and potato dextrose agars. The growth of D. filum on these media is illustrated in Figure 2, A and B, respectively. Some of the morphological features of D. filum grown for several days on lima bean agar are depicted in Figure 3. Not only was Darluca isolated from field collections of the fungus in sori of Puccinia sorghi and other rusts (1), but also studies of possible wide rust host spectra for each isolation, were made (2).

FIGURE 1. Pycnidia of Darluca filum in a sorus of Puccinia sorghi. (From Bull. Torrey Bot. Club. 61, 1934)

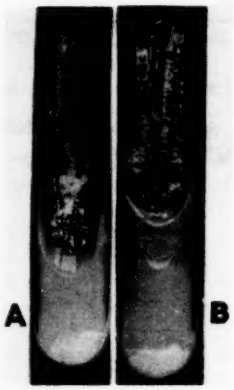

FIGURE 2. Growth of Darluca filum isolated from naturally-infected Puccinia sorghi on lima bean agar (A) and potato dextrose agar (B). (From Proc. Pennsylvania Acad. Sci. Vol. 7. 1933).

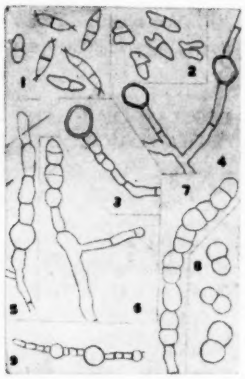

FIGURE 3. Morphological features of Darluca filum grown on lima bean agar: 1, macroconidia; 2, aberrant macroconidia; 3, 4, Chlamydospore-like hyphal cells; 5, 6, 7 and 8, oidia-like cells; 9, differentiated hyphal cells probably a preliminary stage to chlamydospore formation. (From Proc. Pennsylvania Acad. Sci. Vol. 7, 1933).

Isolations of D. filum from sori of Puccinia sorghi were able to parasitize numerous other rusts, among which were: Kuehneola uredinis (Link) Arth., Puccinia acetosae (Schum.) Koern., P. anemones-virginianae Schw., P. antirrhini D. & H., P. circaeae Pers., P. clematidis Lagerh. (= P. rubigo-vera (DC.) Wint.), P. hieracii (Schum.) Mart., P. menthae Pers., Uromyces appendiculatus Fries (= U. phaseoli (Pers.) Wint.), and U. polygoni (Pers.) Fckl. Also it was found (2) that isolations of D. filum from Puccinia sorghi and certain other rusts, would readily re-infect cultures of Puccinia sorghi on Zea mays L. in the greenhouse. Type herbarium preparations from such infections have been reposited in the cryptogamic herbarium of The Pennsylvania State College, State College, Pennsylvania. (Table 1).

In addition to the usual macropycnidia and macroconidia, micropycnidia with microconidia were observed from time to time in certain field collections of Darluca (Eudarluca), particularly in rust sori occurring on plants of the Family Gramineae.

Table 1. Herbarium materials of rusts infected with cultures of Darluca filum isolated from field collections of Puccinia sorghi and other rusts. Materials deposited in the herbarium of The Pennsylvania State College, State College

Penn State Herbarium numbers	Darluca filum isolated from naturally-infected field materials of	Isolation numbers	Inoculated in greenhouse onto
12732-A	Puccinia sorghi on Zea mays	1	Puccinia sorghi on Zea mays
8133-C	Puccinia peckii (= P. extensicola oenotherae) on Carex sp.	12-2	" " "
11133-A	Uromyces fallens (= U. trifolii fallens) on Trifolium pratense	4	" " "
11833-B	Puccinia hibisciata (= P. schedonnardi) on Muhlenbergia schreberi	5	" " "
121632	Uromyces silphii on Juncus sp. (= J. tenuis ?) (Pa.)	3	" " "
12533-A	Puccinia poculiformis (= P. graminis) on Phleum pratense	2	" " "
21033-B	Puccinia violae on Viola sp.	6	" " "
22033-C	Uromyces polygoni on Polygonum aviculare	7	" " "
42133-C	Uromyces silphii on Juncus sp. (= J. tenuis ?) (W. Va.)	3B*	" " "
52233-A	Uromyces junci-effusi on Juncus effusus	8	" " "
72033-A	Puccinia obscura on Juncoides campestre (= Luzula sp.)	11-1	" " "

*Material for this isolation was kindly furnished by Dr. C. R. Orton, West Virginia Agricultural Experiment Station. On various synthetic media the growth of this isolation was quite variable when compared with isolation 3 from the same rust host but collected in Pennsylvania.

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OAK WILT FOUND AFFECTING CHESTNUT OAK IN PENNSYLVANIA

Charles L. Fergus

Large numbers of chestnut oak, Quercus montana Willd. (Q. prinus L.), have been dying and exhibiting branch dieback for the past several years in Pennsylvania. Examination of the affected trees usually revealed the presence in large numbers of species of scale, Asterolecanium spp. Several different species are involved, but most often A. variolosum, the pit-making oak scale was present. In some cases, on the dieback branches, a fungus identified as Sphaeropsis sp. was collected.

With the discovery of oak wilt in Pennsylvania in 1950 by Fergus and Morris[1] it was suspected that some of these chestnut oaks might have been affected by oak wilt. A large number of collections of diseased chestnut oaks have been examined since then and culture isolations attempted. Many specimens were sent in by county farm agents and extension pathologists. None of these yielded Chalara quercina Henry.

In August of this year a specimen was collected from a suspected chestnut oak in Perry County near East Waterford, Pennsylvania. Isolations from this tree yielded pure cultures of the oak wilt fungus.

In a personal communication Mr. W. L. Yount, of the Pennsylvania Department of Agriculture, informed me of his successful isolations from two chestnut oaks located in two separate areas in Bedford County, Pennsylvania in July and August, 1952.

At the Oak Wilt Conference section of the annual meetings of The American Phytopathological Society, held in September at Cornell University, Dr. R. P. True of the University of West Virginia and Mr. L. O. Weaver of the University of Maryland reported the discovery of oak wilt on chestnut oak in West Virginia and Maryland. Although Dietz and Young[2] had shown this species to be susceptible to oak wilt by artificial inoculations, these are apparently the first reports of chestnut oak being affected in nature by oak wilt, thus extending the known natural host range of the disease.

Chestnut, or rock, oak is a commercially important tree in Pennsylvania, particularly on good soils. Its wood is valuable and may be used for the same purposes as the wood of white oak. It is common to the mountainous regions of Pennsylvania, occurring on dry hillsides and rocky ridges. Because of its ability to grow on dry, sterile soils and rocky ridges it serves to prevent soil erosion of areas too poor for the growth of other tree species. If chestnut oak were lost the possibility of increased soil erosion and flash floods would be increased considerably.

DEPARTMENT OF BOTANY, THE PENNSYLVANIA STATE COLLEGE, STATE COLLEGE, PENNSYLVANIA

AN UNUSUAL OCCURRENCE OF OAK WILT IN PENNSYLVANIA

T. L. Guyton

In checking on the sites of oak wilt (Chalara quercina Henry) infections in Pennsylvania, Mr. Arthur R. Jeffery, an inspector with the Pennsylvania Department of Agriculture, observed a line of dead oak trees, mostly red oaks, extending along a trail blazed for the purpose of locating a logging road. This plot of timber land is located in Bedford County near Manns Choice. The blazes were made on small trees ranging up to about six inches in diameter and extended for a distance of possibly one-half mile. The owner of the tract said the blazing was done the last week of April 1952. More than three-fourths of the blazed oak trees were infected by oak wilt and were dead by September 17. The nearest known trees which were thought to have died from oak wilt in 1950 or 1951 are about a quarter of a mile away.

BUREAU OF PLANT INDUSTRY, PENNSYLVANIA DEPARTMENT OF AGRICULTURE, HARRISBURG

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[1] Fergus, C. L., and C. L. Morris. 1950. Oak wilt in Pennsylvania. Plant Dis. Reptr. 34: 291.
[2] Dietz, S. M., and R. A. Young. 1948. Oak wilt. A serious disease in Iowa. Iowa Agr. Exp. Sta. Bul. P 91.

THE SUGARCANE DISEASE SITUATION IN PERU

E. V. Abbott and J. P. Martin

A survey of sugarcane diseases in Peru, with special reference to a new disease now known as Sclerospora disease, was made by the writers during July, 1952, under the auspices of the Comite Productores de Azucar del Peru. Visits were made to a majority of the large haciendas in the northern coastal valleys where most of the sugar of Peru is produced. P.O.J. 2878 is the principal commercial variety of sugarcane, constituting approximately 90 percent of the acreage. The remaining 10 percent is made up principally of the two Hawaiian canes, H. 32-8560 and H. 37-1933, with a locally produced variety, Azul de Casa Grande, being increased on a commercial scale on some plantations.

Sclerospora Disease

The principal purpose of the survey was to determine the distribution and importance of a new disease of sugarcane first observed in the country in April, 1950, and which, following an earlier visit to Peru by one of us (Abbott) in December, 1951, was identified as caused by a species of Sclerospora. It has been termed Sclerospora disease by the pathologists of the Bureau of Sugar Experiment Stations, Queensland, Australia, where the same or a similar disease occurs. The species of Sclerospora involved has not been definitely determined. One or more cases of the disease were observed on six of the nine plantations visited, but in no instance did the infection exceed 1 percent. It was found affecting each of the varieties mentioned above.

Symptoms: Affected plants are severely stunted and usually manifest excessive tillering at the ground level. The leaves become thickened, remain erect, and show a leaf mottling somewhat similar to mosaic disease. Their margins may become wavy in outline, resembling a corn leaf. The dwarfed or diseased plants are in marked contrast with healthy plants in size, color, and general appearance and because of these differences are readily recognized in young plant and ratoon crops. Diseased plants produce little or no millable cane.

Stem galls frequently appear on diseased plants at or near the ground level and seem to be definitely associated with the disease. In some instances the galls vary greatly in size and shape and at times develop into adventitious buds which in turn give rise to shoot proliferation. In some respects the galls differ from those induced with insect extracts as demonstrated in Hawaii by Martin. No mention of galls with the disease has been made in Australia; otherwise the gross symptoms of the two diseases are similar.

Possible Origin of the Disease in Peru: There has been considerable interest in and speculation with respect to the origin of the disease in Peru, and the opinion has been expressed by some that since the disease was first observed in the varieties recently introduced from Hawaii, it was introduced with them. After a study of the situation we found no evidence that the disease was introduced into Peru on the Hawaiian canes. In the first place, Sclerospora disease is not known in Hawaii. Furthermore, the disease is not present on the original plantings of the Hawaiian canes made in Peru, which are still in existence. Also the disease has been found in fields of P.O.J. 2878 that have had no direct contact with the Hawaiian canes. Our observations indicate that the disease has been present on P.O.J. 2878 for some time, but because of the resistance of this variety and consequent low incidence of the disease, it did not attract particular attention. Since certain species of Sclerospora attack a wide range of grass hosts, it is possible that the disease is native to some grasses in Peru, or has been present for some time on another cultivated grass, and has been able to adapt itself to sugarcane.

Economic Importance: The disease has been known to occur in Peru for almost two years and according to some plantation men for a much longer period. Transmission of the disease under field conditions by oospores has not been rapid. Transmission of the disease by cuttings has been principally responsible for its spread from field to field and locality to locality. Transmission of the disease by this method can be controlled. To date economic losses from the disease have not been serious. In view of the past history and nature of the disease economic losses in the future should not be of major importance provided field control measures are adopted.

Control: Control measures that can be recommended include roguing and destroying diseased plants, selection of disease-free planting material, maintaining optimum drainage condi-

tions, and planting resistant varieties. P.O.J. 2878 is resistant to Sclerospora disease, H. 32-8560 is susceptible, and H. 37-1933 is intermediate.

Other Sugarcane Diseases Observed in Peru

Other sugarcane diseases observed during the survey are as follows: Mottled stripe (Xanthomonas rubrisubalbicans), eye spot (Helminthosporium sacchari), red rot (Physalospora tucumanensis), sheath rot (Cytospora sacchari), sooty mold (caused by an unidentified fungus), a leaf spot resembling brown stripe (Cochliobolus stenospilus), mosaic (virus), ratoon chlorosis (nutritional), leaf variegation (believed to be genetic in origin), and chlorotic leaf blotch and cluster stool, which are of unknown origin.

UNITED STATES DEPARTMENT OF AGRICULTURE, HOUMA, LOUISIANA: AND EXPERIMENT STATION, HAWAIIAN SUGAR PLANTERS' ASSOCIATION, HONOLULU, HAWAII.

REDUCTION OF CHERRY DECAY BY WASHING

C. E. Yarwood and H. T. Harvey

A grower reported to the writers that cherries washed with water after picking kept considerably longer than unwashed cherries. As no record of such a treatment was found in the literature examined, the treatment was tested experimentally.

Fruits with attached pedicels and showing no evidence of decay were used within nine hours of picking. There was an average of 170 fruits per treatment for each collection. Washing was by agitating the fruits gently for about 10 seconds in about 3 times their weight of water in 4 successive washes. In most trials 0.1% Triton B1956 (phthalic glyceryl alkyd resin of Rohm and Haas Co.) was used as a detergent in the first two washes. This resin is slightly fungicidal per se, but since some reduction in decay resulted from washing in water without B1956, the action of the B1956 is considered to be primarily due to its detergent qualities. After the final wash an attempt was made to dry the fruit by blowing a strong blast of air over the fruit, but drying was not complete. The washed and unwashed fruit was stored in the laboratory at about 20° C. in open or sealed jars or in unsealed casserole dishes. The percentage decay was greater in sealed than in open containers but the effect of washing was apparently similar for all types of containers.

At six or seven days after start of test each fruit was classified as sound if it had no rotted areas or patches of fungus growth. The few cracked fruits were presumed to be due to the absorption of water by the treated but incompletely dried fruits. The principal fungi present and the only ones that were diagnosed were species of Botrytis, Monilinia, and Penicillium.

In the trial of June 5 with Lambert cherries from Mountain View, California, 29 percent of the untreated fruit, 43 percent of the fruit washed with tap water only, and 55 percent of the fruit washed with B1956 and tap water were sound after seven days. In the trial of June 29 with Royal Ann cherries from Mountain View, 19 percent of the untreated fruits and 83 percent of fruits washed with B1956 and tap water were sound after six days. In the test of July 7 with Royal Ann cherries from Occidental, 67 percent of the untreated fruit and 78 percent of the fruit washed with B1956 and tap water were sound after seven days.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

WINTER INJURY TO TREES IN ILLINOIS

Paul F. Hoffman

Injury to trees and shrubs was observed extensively in Illinois this year. The injury is attributed, mainly, to a sudden severe freeze which occurred in early November, 1951. This premature hard freeze damaged growing tissues of many varieties of ornamental plants. Numerous inquiries were received during the 1952 growing season concerning this dying of otherwise healthy plants. Trees most severely injured were weeping willow, Chinese elm, and soft maple. Trees less severely injured were pussy willow, black willow, ash, mountain ash, hawthorne, American elm, poplar, mulberry, ginkgo, pin oak, osage orange, and numerous fruit trees.

Injury developed in several patterns. In one pattern, killing of the bark at the base of the tree resulted in death of affected plants. This type of injury occurred primarily on Chinese elm and weeping willow early in the growing season. In another, more common pattern, injury occurred as foliage wilt and dieback of twigs and branches, especially upper branches. Early season symptoms were dying of buds preceding or shortly following break of dormancy. Trees that started to leaf out in an apparently normal manner frequently showed stunted, chlorotic foliage and reduced shoot growth followed by defoliation and twig dieback. Late season symptoms were yellowing and dropping of leaves accompanied by twig dieback. Drought aggravated the late season symptoms and further weakened previously damaged plants.

The injury to weeping willows and Chinese elms was most conspicuous and widespread. These are fast growing trees used abundantly in ornamental plantings. They prolong growth in the fall and begin growth early in the spring, characteristics rendering them particularly susceptible to unseasonable freezes.

November, 1951, was the coldest November in Illinois since the origination of state-wide records in 1890[1]. The average temperature for the State during this month was 35.5° F. The

[1]United States Weather Bureau. Climatological Date for Central United States. Volume 56. Nos. 10 and 11. 1951.

lowest temperature of the month was -2° F., reported at Freeport on November 5. On November 3 no weather station in the State reported a minimum temperature above 20° F. and most stations reported temperatures in the vicinity of 10° F. Snowfall in November was 7.9 inches, which exceeded previous records. Heavy snows fell on the 6th and 7th of November.

The sudden cold and snow followed a period of above-normal temperatures in October. While one station reported a low reading of 24° F., there were no widespread hard freezes. Only during the last three days of October did stations, in general, in the northern third of the State record temperatures as low as the upper twenties.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA, ILLINOIS

DISEASES OF TOMATO, PEPPER, SQUASH, OKRA AND COTTON OBSERVED IN WEST TEXAS

P. A. Young

Farm crops were surveyed for diseases near Lubbock, Littlefield, Muleshoe, and New Deal in Texas and Lovington in New Mexico from July 30 to August 1, 1952. They included large irrigated fields of tomatoes that represented about 500 acres near Lubbock and about 1000 acres near Lovington.

The abnormal tomato plants in the fields showed typical symptoms of the curly-top virus disease which apparently killed or damaged 15 to 85 percent of the plants. No other disease was found in the tomatoes. Many of the plants with curly-top symptoms had died while they were very small, according to farmers' statements. Many of the brown, dead plants remained in the tomato rows. In addition to the symptoms of curly top that are described in literature, the following should be mentioned. The first symptom in tomato plants about two feet tall was yellowing of one or a few leaflets. Purpling of the hypophyllous veins of a few slightly rolled upper leaflets was a doubtful early symptom. A grayish discoloration of the leaves was associated with stopping of the growth of tomatoes by curly top. The plants with leathery leaves made an abnormal paper-like rustle when they were shaken. Such leaves wilted and became soft before they died.

Weed hosts are an important source of the curly-top virus. Hence it was important to notice, in two tomato fields, that a few of the small Amaranthus plants showed some brittle, rolled leaves as symptoms of curly top.

Pepper curly top: Curly top was the only apparent cause of the killing or spoiling of about 80 percent of the bell pepper plants in a three-acre field near Lovington. Most of the plants had died while they were very small, according to the owner. The remaining diseased plants showed typical symptoms of curly top, including failure to set fruit. Instead of yellowing, some of the plants had clusters of flowers in their tops, and the leaves were cup-shaped with irregular margins. Larger pepper plants in a large field near Littlefield showed about 1 percent of the plants with typical symptoms of curly top.

Squash curly top: A field of straight-neck squash with ripening fruits was examined near New Deal. About 10 percent of the plants showed typical symptoms of curly top, including white to yellow discoloration and leathery texture of the leaves. The plants with curly-top symptoms produced few marketable fruits despite continued stem growth and blooming that had ceased in the normal plants.

Okra wilt: A field of greenpod okra showed about 10 percent of the plants with severe dwarfing of the stems and dying of many of the leaves. Sections of the stems and leaf petioles showed prominent blackening of the xylem tissues, indicating wilt caused by Verticillium, or possibly by Fusarium. The soil was reported to be alkaline.

Cotton angular leaf spot (Xanthomonas malvacearum): This bacterial disease of cotton was severe in two fields near Lovington.

TOMATO DISEASE LABORATORY, TEXAS AGRICULTURAL EXPERIMENT STATION, JACKSONVILLE, TEXAS

NEW RECORDS OF PLANT DISEASES

TAKE-ALL OF WHEAT IN GEORGIA

By H. B. Harris, U. R. Gore, and E. S. Luttrell

During the spring of 1952 an infection of take-all[1] developed in a planting of wheat at Experiment, Georgia. Although take-all has been reported along the Atlantic Seaboard from New York to North Carolina[2], it is uncommon as far south as North Carolina. Its occurrence in Georgia represents a significant southward extension in the known range of this disease.

The disease occurred in a wheat fertilizer test which included 126 plots representing 6 replications each of 21 fertilizer, lime, and cover crop treatments. Several infected areas were found in this test on April 2, 1952, just before the wheat began to head. The most conspicuous area extended across two plots and measured 30 by 10 feet. The infected plants were more or less stunted and yellowed. Those in the center of the area were only about 8 inches tall, and some were dead. Infected plants showed characteristic symptoms of take-all, a blackening of the base of the plants produced by mats of dark mycelium growing beneath the leaf sheaths. Perithecia of the causal fungus were present in the basal leaf sheaths of the dead plants.

Additional areas of infection became apparent during April and May. On June 2, when the plots were harvested, notes were taken on the percentage of infection in each plot. The infected areas were distributed at random over the field. There was no evident correlation between degree of infection and fertilizer, lime, or cover crop treatments. Only 3 percent of the plants in the entire field were infected. Although there were numerous areas of slight infection, infections, in excess of 20 percent occurred in only eight plots. In the most seriously affected plot 59 percent of the stand was infected. The yield from this plot was reduced by 71 percent as compared with the average yield of five noninfected plots in the same treatment.

There was nothing to indicate the means by which take-all was introduced into this field or the factors favoring its development. Rainfall and temperatures during the spring of 1952 were close to the averages. One fact of significance is that the field in which the disease occurred had been planted in wheat continuously for five years. The causal fungus may have been present for several years and gradually developed to the point of causing noticeable damage.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

[1]Caused by Gaeumannomyces graminis (Sacc.) v. Arx & Olivier (= Ophiobolus graminis Sacc.) Arx, J. A. von, and Dorothea L. Olivier. 1952. The taxonomy of Ophiobolus graminis Sacc. Trans. British Mycol. Soc. 35: 29-33.

[2]Weiss, Freeman. 1950. Index of plant diseases in the United States. Part III. Gramineae. U.S.D.A. Plant Dis. Survey Special Pub. No. 1, Part 3.

FUSARIUM WILT IN FLUE-CURED TOBACCO IN VIRGINIA

By Wilbert A. Jenkins

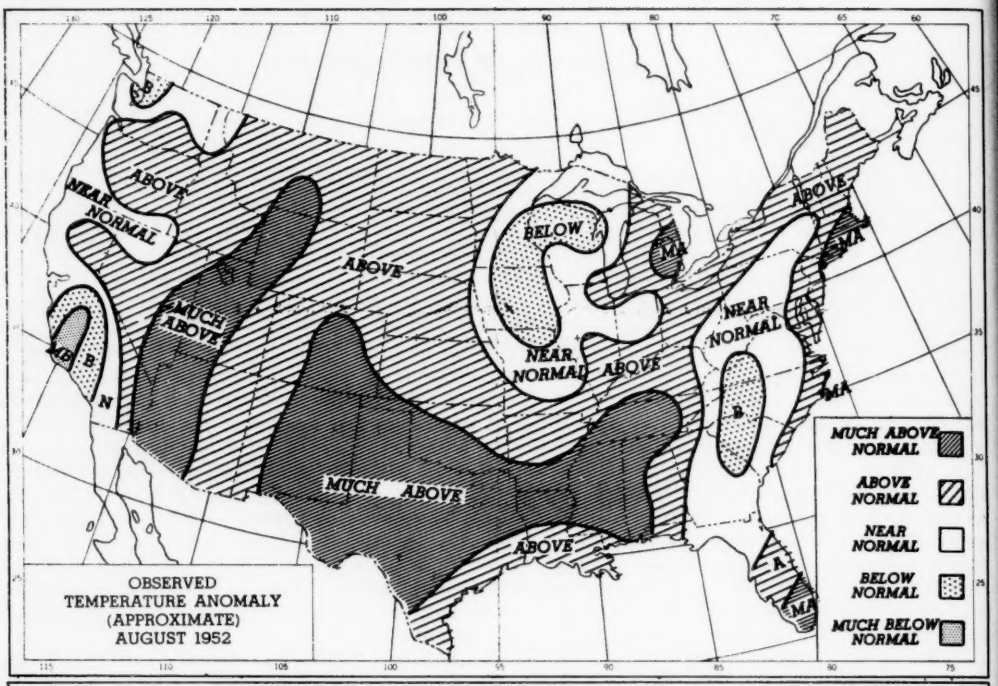
Fusarium wilt (F. oxysporum f. nicotianae) was discovered August 14, 1952 in a single field of flue-cured tobacco in Pittsylvania County. A considerable section of a several-acre field had been killed out by wilt when first seen and evidence of spotting of the disease in several other sections of the field was noted. Wilt, in this field, was accompanied by severe root knot (Meloidogyne sp.) infestation. Classic symptoms, external and internal, were present on all plants examined and specimens have been sent to Dr. G. M. Armstrong, Clemson College, for typing of the pathogen.

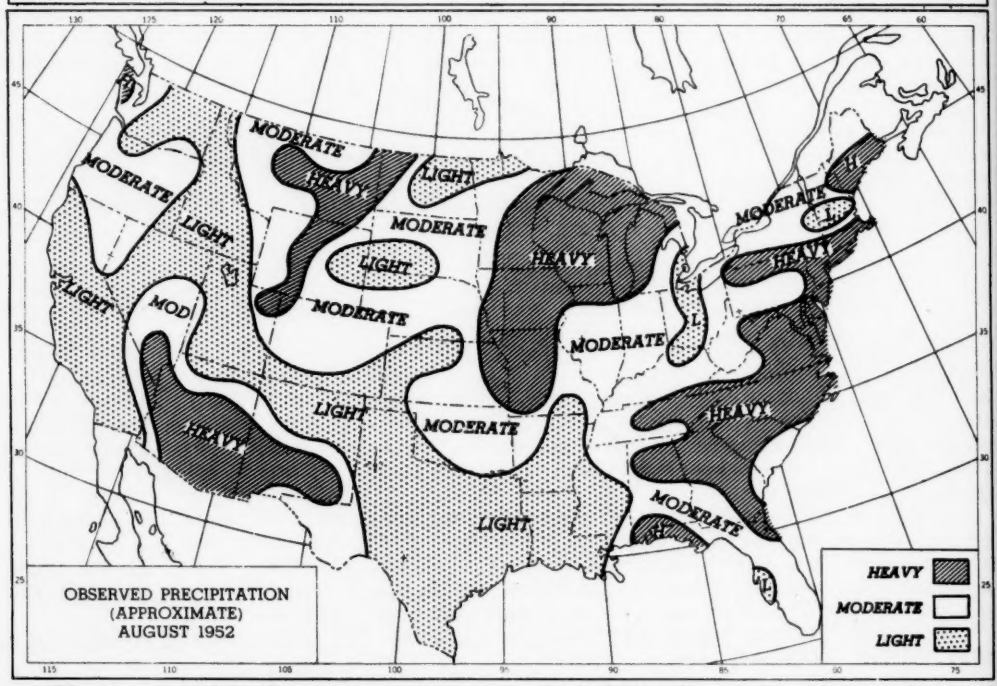
Fusarium wilt has been reported previously from southwestern Virginia (Lee County) in Burley tobacco, but this is the first time it has been noted in flue-cured tobacco in Virginia. No information is at hand to explain the present infestation, though it is known that the owner of the farm has tobacco interests in the Whiteville-Chadbourn area of North Carolina and it is possible that the infestation may have developed from plants from that area.

VIRGINIA AGRICULTURAL EXPERIMENT STATION, TOBACCO DISEASE RESEARCH AND SEED FARM, CHATHAM

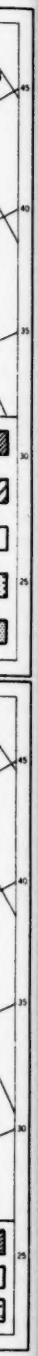
A CORRECTION

We regret that the article by Folke Johnson "Black raspberry mosaic in Western Washington" was omitted from the contents for the June 15 issue, in which it appeared on pages 239-240.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.



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